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Activity-guided isolation and characterisation of the anti-complement principles of a methanol extract of the gum of Boswellia serrata

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Frankincense, the gum resin of *Boswellia serrata*, is used in traditional medicine for the treatment of inflammatory disorders. For extracts of the gum, hepatoprotective and anti-complement activities and inhibition of leukotriene synthesis have been reported (1). So far, only crude extracts of *Boswellia* were tested for their effects on complement activity (2). The present paper records the isolation and characterisation of the active principles of a methanol extract of the gum of *Boswellia* serrata as well as a study on their mechanism of action.

Guided by the activity on the classical pathway of human complement, 4 compounds with a potent anti-complement activity were identified: α -boswellic acid, β -boswellic acid, acetyl- β -boswellic acid and acetyl- α -boswellic. Of these active principles, acetyl- β -boswellic acid showed the highest inhibitory activity (IC $_{50}$ 4.6 µg/ml). This activity was selective for the classical pathway, no activity was observed towards the alternative pathway. Furthermore, the inhibitory activity was not affected by raising the Ca $^{2+}$ and Mg $^{2+}$ concentration.

Detailed mechanistic studies revealed that acetyl-β-boswellic acid acts at the level of complement component C2.

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References: 1. Ammon, H.P.T. et al. (1991) Planta Med. 57: 203-207. 2. Kapil, A., Moza, M. (1992) Int. J. Immunopharmac. 14: 1139-1143.

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Action mechanism of Croton cajucara Benth in the treatment of gastric ulcer detected by RT-PCR and somatostatin dosage in normal and malnourished rats

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Aim: Analyze the perfomance the anti-ulcerogenic activity of the *Croton cajucara* essential oil in acetic acid and ethanol induced gastric ulcer in normal and malnourished rats, and the "repair" process involved with these ulcers. Serum somatostatin (SMT) was also examined.

Methods: Acetic acid and ethanol-induced gastric ulcer were assayed according to the method of Takagi et al. (1) and Morimoto et al. (2), respectively. The repair process involved with these ulcers (growth factors by RT-PCR and prostaglandin PGE₂) was performed according to the Konturek et al. (3) and Curtis et al. (4) methods, respectively. Serum SMT was assayed by Karmely et al. (5).

Results: The dates shown that a single oral administration of essential oil (100 mg/kg) accelerated the healing of chronic gastric ulcer (49,37% and 69,82%) in both groups when compared to the control group (P<0.001). We also observed that this drug test induced a significantly increase in PGE2 production by glandular cells (50% compared to control) in both groups. In normal rats exposed to acetic acid, growth factors how EGF was increased several-fold in the gastric lumen compared with the value measured in intact animals. The serum somatostatin value of malnourished animals was increased with or without the use of essential oil (P<0.001), and the normal animals had high value of SMT with the essential oil treatment.

Conclusion: The protective and curative effect of essential oil of *C. cajucara* bark on induced gastric lesions was effective in normal and malnourished rats by specific action mechanisms.

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References: 1. Takagi et al. (1969) Jap. J. Pharm. 19: 418-426. **2.** Morimoto et al. (1991) Jap. J. Pharm. 57: 495-505. **3.** Konturek et al. (1990) Gastroenterol. Clinical American 19: 41-65. **4.** Curtis et al. (1995) Canadian J. Physiol. Pharmacol. 73: 130-134. **5.** Karmeli et al. (1994) Gastroenterology 10: 1206-1216.